Diagnosis of Respiratory Distress Syndrome (Hyaline Membrane Disease) by Amniocentesis

Prevention of respiratory distress syndrome (RDS) of the newborn (hyaline membrane disease) is now possible through recent research work on the various surface active phospholipids in lungs. As fetal lungs mature lecithin appears in significant amounts in addition to the less effective, earlier appearing sphingomyelin. The ability of the lung to remain expanded after birth is directly related to the amount of lecithin present. These phospholipids can readily be found in the amniotic fluid, thus the ratio of lecithin to sphingomyelin (L:S) may be determined in a test tube thin layer chromatogram after appropriate extraction and simple purification. The relative quantities are roughly estimated measuring height and width of the spots on the developed chromatograms. If the L:S ratio is less than 1.5:1 the risk of respiratory distress syndrome is very high. If the ratio is greater than 1.8:1 the risk is very low. By waiting in elective situations until the ratio is favorable, RDS may be prevented. In the event that delivery cannot be postponed the physician is forewarned and may initiate appropriate steps before birth.

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Mechanisms of Blood Clotting

Clotting factors can be classified into three groups: (1) thrombin-sensitive factors: viz., fibrinogen and factors V, VIII and XIII; (2) vitamin K-dependent: viz., prothrombin, factors VII, IX and X; (3) contact factors: viz., factors XI, XII and possibly the recently described Fletcher factor. The clotting sequence is triggered off by a reaction involving Hageman factor (XII); the activated Hageman factor then reacts with factor XI converting it to an active form, XIa, which in turn activates factor IX. In the presence of calcium ions the activated factor IX reacts with factor VIII and phospholipids released from platelets to form a complex which converts factor X to its activated form, Xa. The Xa in turn reacts with factor V, platelet phospholipids and calcium to form another complex which converts prothrombin to thrombin. This enzyme converts fibrinogen to fibrin monomer by splitting off two small polypeptides from the molecule and the fibrin monomer then polymerizes to form a fibrin clot. Finally the fibrin clot is rendered more stable and urea insoluble by factor XIII following its activation by thrombin. The first traces of thrombin potentiate factors V and VIII but as the thrombin level increases these factors are destroyed, clotting is decelerated and the thrombin already formed is neutralized by various antithrombins. In the presence of tissue juices the above pathway (referred to as the *intrinsic* system) is short-circuited; a protein factor and certain phospholipids in the tissue react with factor VII in the presence of calcium to activate factor X directly. The subsequent events in this, the *extrinsic* system, are the same as in the intrinsic system.

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Practical Application of the Use of Immunofluorescence in Renal Biopsy

With the availability of the cryostat and monospecific fluorescein-labeled antisera from commercial sources, fluorescent microscopy can become part of the routine evaluation of a renal biopsy. Generally the technique is the direct method, using antisera against human IgG, complement, or fibrinogen; no consistent staining having been found with antisera against other serum proteins.